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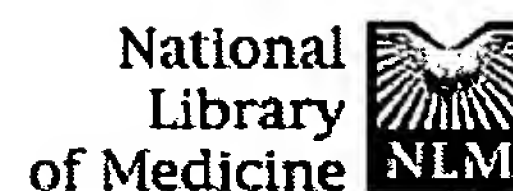
Department of Medical Biochemistry, University of Calgary, Alberta, Canada.

The nematode *Caenorhabditis elegans* was transformed with constructs containing upstream deletions of the gut-specific *ges-1* carboxylesterase gene. With particular deletions, *ges-1* was expressed, not as normally in the gut, but rather in muscle cells of the pharynx (which belong to a sister lineage of the gut) or in body wall muscle and hypodermal cells (which belong to a cousin lineage of the gut). These observations suggest that gut-specific gene expression in *C. elegans* involves not only gut-specific activators but also multiple repressors that are present in particular nongut lineages.

PMID: 2020855 [PubMed - indexed for MEDLINE]

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☐ 1: Proc Natl Acad Sci U S A 1986 Dec;83  
(24):9551-5

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## Direct introduction of genes into rats and expression of the genes.

Benvenisty N, Reshef L.

A method of introducing actively expressed genes into intact mammals is described. DNA precipitated with calcium phosphate has been injected intraperitoneally into newborn rats. The injected genes have been taken up and expressed by the animal tissues. To examine the generality of the method we have injected newborn rats with the chloramphenicol acetyltransferase prokaryotic gene fused with various viral and cellular gene promoters and the gene for hepatitis B surface antigen, and we observed appearance of chloramphenicol acetyltransferase activity and hepatitis B surface antigen in liver and spleen. In addition, administration of genes coding for hormones (insulin or growth hormone) resulted in their expression.

PMID: 3540943 [PubMed - indexed for MEDLINE]

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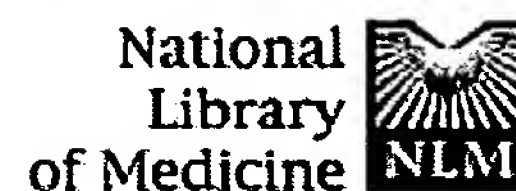
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☐ 1: Mol Biochem Parasitol 2001 Apr 6;113  
(2):223-32

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## Functional analysis of leucine aminopeptidase in *Caenorhabditis elegans*.

**Joshua GW.**

London School of Hygiene and Tropical Medicine, Department of Infectious and Tropical Diseases, Keppel Street, WC1E 7HT, London, UK. g.joshua@lshtm.ac.uk

To investigate the function of the enzyme leucine aminopeptidase in nematodes, a *Caenorhabditis elegans* leucine aminopeptidase gene identified in the genome sequence was functionally analysed by transfection of a leucine aminopeptidase beta-galactosidase reporter construct and characterisation of a null mutant. The leucine aminopeptidase transgene is expressed along the length of the gut, and immunolocalisation shows the enzyme in the buccal cavity, pharynx, anterior gut and rectum. It is constitutively expressed as seen by analysis of cDNAs constructed from mRNAs of nematodes taken at 2 h intervals through the life-cycle; and by western blot analysis of protein from the same set of nematodes. Leucine aminopeptidase null mutants had a slower growth rate and delayed onset of egg-laying. We suggest that in *C. elegans*, leucine aminopeptidase is a digestive enzyme.

PMID: 11295176 [PubMed - indexed for MEDLINE]

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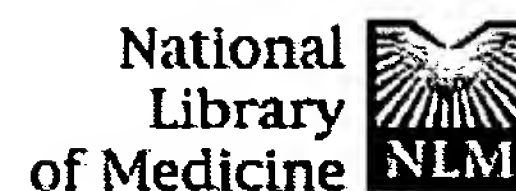
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- ☐ 1: Dev Biol 1996 Sep 15;178(2):276-88 Related Articles, **NEW Books**, LinkOut



## Modulation of gene expression in the embryonic digestive tract of *C. elegans*.

**Fukushige T, Schroeder DF, Allen FL, Goszczynski B, McGhee JD.**

Department of Medical Biochemistry, University of Calgary, Alberta, Canada.

The *Caenorhabditis elegans* digestive tract is composed of four distinct modules derived from separate cell lineages: anterior pharynx from the ABa lineage, posterior pharynx from the MS lineage, gut from the E lineage, and rectum from the ABp lineage. The *C. elegans* gut esterase gene (*ges-1*) is normally expressed in the embryonic gut or E lineage. However, expression *ges-1* can be switched into cells of the embryonic pharynx and tail by virtue of deleting a tandem pair of WGATAR sites in the *ges-1* promoter. Here, we use both laser ablation experiments and genetic analysis to show that cells expressing the WGATAR-deleted *ges-1* transgene belong to all three nongut lineages of the digestive tract: ABa, MS, and ABp. We also show that the molecular size and spatial distribution of *ges-1* mRNA transcripts produced by either the WGATAR-deleted *ges-1* transgene or the undeleted *ges-1* control transgene appear correctly regulated, suggesting that the spatial switch in *ges-1* expression occurs at the level of transcription initiation. We further show that both the WGATAR-deleted and the undeleted *ges-1* transgenes respond appropriately to mutations in a series of maternal effect genes (*skn-1*, *mex-1*, *pie-1*, and *pop-1*) that alter early blastomere fate. Moreover, the pharynx/tail expression of the WGATAR-deleted *ges-1* transgene is abolished by mutations in the zygotic gene *pha-4*. Finally, we use imprecise transposon excision to produce two independent *C. elegans* strains with 1- to 2-kb deletions that remove the tandem WGATAR sites from the promoter of the endogenous chromosomal *ges-1* gene: in both of these strains, *ges-1* is not expressed in the embryonic gut but is expressed in cells of the embryonic pharynx; pharynx expression is weak but incontrovertible. Overall, our results validate previous transgenic analysis of *ges-1* control and show further that *ges-1* appears to be

regulated in a system-specific, rather than a lineage-specific, manner. The multiple facets of ges-1 expression provide an opportunity to investigate how a multicomponent organ system such as the digestive tract is established from diverse cell lineages.

PMID: 8812129 [PubMed - indexed for MEDLINE]

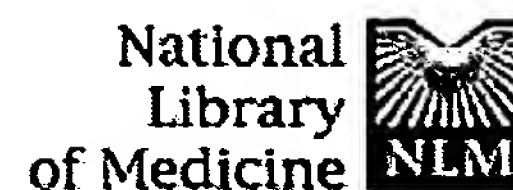
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☐ 1: J Mol Biol 1993 Feb 20;229  
(4):890-908

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## The gut esterase gene (ges-1) from the nematodes *Caenorhabditis elegans* and *Caenorhabditis briggsae*.

Kennedy BP, Aamodt EJ, Allen FL, Chung MA, Heschl MF,  
McGhee JD.

Department of Medical Biochemistry, University of Calgary, Alberta,  
Canada.

The ges-1 gene codes for a non-specific carboxylesterase that is normally expressed only in the intestine of the nematode *Caenorhabditis elegans*. In the current paper, we describe the cloning and characterization of the ges-1 gene from *C. elegans*, as well as the homologous gene from the nematode *Caenorhabditis briggsae*. The ges-1 esterases from the two nematodes are 83% identical at the amino acid level and contain regions of significant similarity to insect and mammalian esterases; these conserved regions can be identified with residues believed to be necessary for esterase function. The ges-1 mRNAs from both *C. elegans* and *C. briggsae* are trans-spliced. The coding regions, the codon bias and the splicing signals of the two ges-1 genes are quite similar and most (6/7) of the intron positions are retained precisely. Yet, the flanking sequences of the two ges-1 genes appear to have diverged almost completely. For example, the *C. elegans* ges-1 5'-flanking region (as well as several introns) contains copies of three different SINE-like sequences, previously identified near the hsp-16 genes, near the unc-22 gene and in a repetitive element CeRep-3; none of these elements are found in the *C. briggsae* ges-1 gene. We show that: (1) the *C. elegans* ges-1 gene can be used to transform *C. briggsae*, whereupon expression of the exogenous ges-1 gene is confined to the *C. briggsae* intestine; (2) the ges-1 homologue cloned from *C. briggsae* can be transformed into *C. elegans*, whereupon it is expressed largely in the *C. elegans* intestine; and (3) a 5'-deletion of the *C. elegans* ges-1 gene that we have previously shown to be expressed in the *C. elegans* pharynx is also expressed in the pharynx of *C. briggsae* (either in the presence or absence of vector sequences). These results suggest that the ges-1 gene control circuits have been

maintained between the two nematode species, despite the divergent 5'-flanking sequences of the gene. This raises the question of the evolutionary distance between *C. elegans* and *C. briggsae* and we attempt to estimate the *C. elegans*-*C. briggsae* divergence time by analysing the rate of synonymous substitutions in coding regions of *ges-1* and six other *C. elegans*-*C. briggsae* gene pairs. We propose a new method of analysis, which attempts to remove rate differences found between different genes by extrapolating to zero codon bias.(ABSTRACT TRUNCATED AT 400 WORDS)

PMID: 8445654 [PubMed - indexed for MEDLINE]

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